quickly stained. It is, however, to the fourth Resuscitation Committee (1889–1903), or, to state the matter accurately, to Professor Schäfer, that we owe our most accurate knowledge of the absorption of water by the lungs in the act of drowning. He found that the amount taken into the lungs of dogs varied greatly. The sixth dog of his experimental series took 780 cubic centimetres of water into its lungs during an immersion of five minutes and recovered spontaneously. He found that the air expired in the act of drowning was equal to only a fraction of the water taken into the lungs. A specimen from the collection in the Museum of the Royal College of Surgeons of England of the lungs of a cat drowned in water containing plaster-of-Paris shows that the air-passages from the trachea to the terminal bronchioles are filled with plaster ; near the roots and along the dorsal parts the plaster has even reached the infundibula and air cells. The lungs of drowned persons are large and do not collapse : (1) because their capillaries are overladen with blood ; (2) because the air passages and spaces of the dorsal and deeper parts of the lung are filled with water ; and (3) because the anterior (ventral) and diaphragmatic parts of the lung are distended with air. The appearance of the lungs gave rise to the ancient belief that the drowned and hanged died in the act of inspiration. The parts of the lungs which become emphysematous in the act of drowning are those which are most liable to this condition in common respiratory diseases. I do not propose to discuss now the cause of emphysema, but those interested will find this condition very thoroughly analysed by N. Ph. Tendeloo.[14] The capillary system of the lung is often seriously damaged. How often the froth that exudes from the respiratory passage is blood-stained I cannot discover ; in the records— often very brief—of the Royal Humane Society it is occasionally noted. I had an opportunity of examining the lungs of the walrus which was accidentally drowned in the Zoological Gardens during the present winter. Mr. William Pearson, prosector of the College, poured a solution of plaster-of-Paris into the trachea ; we were surprised to note that in several of the deeper parts of the lungs it had also passed into the smaller veins ; we were able to trace these veins to infundibula full of plaster-of-Paris. On microscopic examination large areas of the lungs were found to be full of blood from rupture of the smaller vessels. At these points of rupture the plaster entered the vessels. I infer that air would also have entered into the veins if artificial resuscitation had been applied. Is it not possible—nay, probable— that some of the cases which died suddenly after being partially restored are killed by air embolism ? Out of six cases of asphyxia in young children Dr. Ivy McKenzie[15] found large pulmonary extravasation of blood in one, and minute extravasations in four. Whichever form of resuscitation be adopted it is important that the condition of the lung be kept in mind.

INFLATION OF THE LUNGS CONTRASTED WITH NATURAL BREATHING.

Inflation of the lung does not produce a pulmonary movement similar to that which takes place either in a natural inspiration or in an inspiration produced by an artificial expansion of the chest. When air is blown into the lungs those parts expand—as J. Hutchinson[16] demonstrated in 1852—which are situated against the most yielding parts of the thoracic cavity. The most yielding wall is (1) the diaphragmatic and (2) the anterior sterno-chondral wall. Hence when the lungs are inflated the chief movement seen is one at the epigastrium. The water-logged parts of the lungs lying against the rigid apical and dorsal walls are expanded to a much less extent. The resistance offered by the thoracic wall to the expansion of the lungs was found by Hutchinson to be very considerable. In two men, just dead, he found that a pressure of 30 millimetres of mercury was necessary to introduce 1000 cubic centimetres (twice the amount necessary for resuscitation), whereas when he removed the lung from the thorax he could introduce 1500 cubic centimetres at a pressure of 10 millimetres of mercury. In one case in which he inflated the lungs while *in situ* he found that they ruptured

when 1430 cubic centimetres had been introduced at a pressure of 37·5 millimetres of mercury. Inflation differs also from natural inspiration in its action on the blood-content of the lung ; whereas inflation diminishes the amount of blood and lymph in the lung by forcing on the blood and lymph along their courses natural inspiration serves to increase the volume of blood and lymph within the lung. The expiratory movement caused by compressing the epigastrium and anterior wall of the sternum does not differ materially from the effect of a natural expiration. Compression applied to the anterior wall of the chest acts chiefly on the anterior part of the lung—the dorsal and apical parts are but slightly affected. The pressure applied serves to empty the lung of air and blood.

TABES DORSALIS WITH UNILATERAL ANÆSTHESIA : A CONTRIBUTION TO THE PATHOGENESIS OF THE DISEASE.

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I. *Introduction.*—It is probable that no disease of the spinal cord has been investigated more fully than tabes dorsalis, but in spite of this there is much divergence among neuro-pathologists as to the nature and starting-point of the tabetic process. All workers are now agreed that there exist in the posterior columns of the spinal cord two systems of fibres : (1) an exogenous system composed of the intra-medullary prolongations of the posterior roots, and (2) an endogenous system of fibres which connect parts of the central nervous system with one another. This latter set of fibres consists of an ascending system, the cornu commissural tract, and of a descending system of fibres composed of the comma tract of Schultze, the posterior bundle of Hoche, the median oval area of Flechsig, and the triangle of Gombault and Phillippe. In early cases of tabes it is the former or exogenous system that is exclusively affected, so that the wasting affects not the posterior columns as a whole but only that part of them consisting of the intramedullary prolongations of the posterior roots. The old theory of the origin of tabes in a syphilitic vascular lesion of the posterior columns may therefore be dismissed, and for the same reason its modern representative, the specific lymphangitis of the posterior columns described by Marie and Gullain,[1] must share the same fate.

There remain, then, two views of the origin of tabes : (1) the radicular theory, that the disease is due to a lesion of the posterior roots outside the spinal cord and that the wasting of the posterior columns is merely the result of a secondary degeneration of their intramedullary terminations ; and (2) the dystrophic theory, that the disease is due to a primary degeneration of the sensory proto-neurons and that the thickening of the membranes, the changes in the vessel walls and the neurogliosis are secondary to the atrophy of the nerve fibres. As Ferrier[2] shows in his most recent contribution to the subject, the radicular theory is still held by the majority of neuro-pathologists. Much research has been devoted to endeavouring to locate the point at which the posterior roots are attacked. Nageotte[3] holds that the anterior and posterior roots are affected by a chronic diffuse meningitis of syphilitic origin between the posterior root ganglion and their point of exit from the dural sac. This portion of their course he terms the "nerf radiculaire." He considers that in this part of their course the anterior and posterior roots are especially liable to inflammation and that a transverse neuritis is set up in them leading to secondary degeneration of the posterior roots. As Ferrier[4] points out, however, the practical escape of the anterior roots is a fatal objection to Nageotte's theory. That the neuritis of the anterior

[14] Studien ueber die Ursachen der Lungen-Krankheiten, Wiesbaden 1902, pp. 470.
[15] Journal of Anatomy and Physiology, 1906, vol. xl , p. 120.
[16] Todd's Cyclopædia of Anatomy and Physiology, 1852, vol. iv., article " Thorax."

[1] Revue Neurologique, 1903, No. 2, p. 49.
[2] Ferrier : Lumleian Lectures on Tabes Dorsalis, THE LANCET, March 31st (p. 881), April 7th (p. 951) and 14th (p. 1017), 1906.
[3] Nageotte : La Presse Médicale, Dec. 10th, 1902, and Jan. 3rd, 1903.
[4] Ferrier : Op. cit.

root should be evanescent while that of the posterior radicle should lead to such profound intramedullary degeneration is unintelligible and is, moreover, opposed to the usual relatively greater affection of the motor than of the sensory fibres, when neuritis attacks mixed nerves. Redlich and Obersteiner [5] maintain that the posterior roots are constricted where they penetrate the pia mater, and that in consequence of meningeal thickening resulting from old syphilitic meningitis the posterior roots become nipped and degeneration of the intramedullary exogenous tracts results. Orr and Rows [6] point out that the posterior roots at their entrance to the spinal cord lose their neurilemma or external sheath, and they consider the posterior roots to be in consequence especially vulnerable at this point of their course.

On the other hand, Thomas and Hauser, [7] after a most careful and complete research, maintain that the essential lesion of tabes is a dystrophy affecting the primary sensory neurons. They consider that the whole sensory neuron is affected both in its peripheral and central terminations, the intramedullary portion being the earliest to show anatomical change. They believe that the degeneration of the nerve fibres is a simple atrophy, that the myelin sheath and the axis cylinder disappear slowly and progressively, and that true Wallerian degeneration is exceptional. Marie [8] was the first to advance the attractive theory that the primary lesion was situated in the cells of the posterior root ganglia, while von Leyden and Goldscheider [9] are led by their work to regard the peripheral nerve endings as the starting-point of the tabetic process.

II. *The nature of the sensory changes in tabes dorsalis.*— For the past six years the writer has been engaged, in conjunction with Dr. Henry Head, in an investigation of the sensory changes associated with lesions of the spinal cord. During this time a number of cases of tabes dorsalis have been investigated of which in a short paper like the present it is impossible to give a detailed account. The following case which came under the writer's own care is published here because, although in some ways unusual, it illustrates several of the conclusions we have reached and because the nature of the lesion was verified by post-mortem examination.

The grouping of sensory impulses in the posterior roots and peripheral nerves has been determined by Head, Rivers, and Sherren, [10] while the sensory changes produced by lesions of the secondary sensory system have been investigated by Head and Thompson. [11] It is clear that the nature of the anæsthesia in tabes dorsalis must be investigated afresh in the light of these researches. Dr. Head has kindly allowed the use of his material for the present paper, which embodies the conclusions arrived at by himself and the writer on the nature of the sensory changes in tabes dorsalis.

In lesions of the posterior roots and the peripheral nerves three areas of defective sensibility can always be marked out : (1) the area of protopathic loss, over which are abolished cutaneous pain and sensibility to heat from temperatures above about 50° C. and to cold from temperatures below about 20° C. ; (2) the area of epicritic loss, over which are absent light touch and cutaneous localisation, tactile discrimination (as determined by the compasses), and discrimination of intermediate degrees of temperature from about 25° C. to about 40° C. ; and (3) the area of loss of deep sensibility over which are abolished tactile pressure, pain produced by excessive pressure, and recognition of passive position at the joints and of active movement of the muscles.

In lesions of the secondary sensory system within the spinal cord it is found that : (1) Painful sensibility and thermal sensibility are often lost together, yet analgesia may exist without thermo-anæsthesia, and sensibility to heat may be abolished without coincident disturbance of that to cold. (2) Painful sensibility is disturbed as a whole. When the skin is analgesic sensibility to the pain of deep pressure is also diminished or absent, although the pressure itself and its gradual increase can be appreciated. Thus impulses

started by cutaneous painful stimulation arriving by the protopathic system and those due to painful pressure arriving by the deep system of nerves become combined and part company from all other sensory impulses that may have travelled with them in their peripheral paths. (3) When sensibility to heat is disturbed in consequence of a lesion of the secondary sensory system it is affected as a whole. There is no separation of the loss to extreme and minor degrees of temperature such as is seen in peripheral nerve lesions. A similar statement holds good for sensibility to cold. (4) Sensibility to light touch and to pressure are affected simultaneously. Thus, impulses started by light tactile stimulation arriving by means of the epicritic fibres and those due to pressure arriving by the deep system of nerves become combined into one group of tactile impulses and part company from the other sensory impulses which have travelled with them in their peripheral paths. (5) Loss of the sense of passive position and movement is independent of the loss of tactile sensibility but is closely associated with inability to discriminate two compass points.

The following figure (Fig. 1), which is purely diagrammatic, may assist in making clear the recombination of sensory impulses at the secondary or spinal level. The protopathic fibre (dotted line) on reaching the spinal cord breaks up around the specific receptors for pain (a), heat (b), and cold (c). The epicritic fibre (continuous line) on reaching the spinal cord breaks up around the specific receptors for heat (b) and cold (c). Its collateral branches are also connected with the specific receptors for tactile impulses (d) and it continues up the whole length of the posterior columns to the specific receptor for impulses of tactile discrimination (e). The fibre of the deep system (broken line) breaks up around the specific receptor for pain impulses (a), around that for tactile impulses (d), and finally passes up the whole length of the posterior column to the specific receptor for impulses of passive position and active moment (e). Its collaterals also arborise around the cells of Clarke's column (f). Impulses from these specific receptors more or less rapidly pass across to the opposite side of the spinal cord. Those for painful and thermal impulses pass across the spinal cord and pass up among the fibres of the antero-lateral tract of Gowers (1). For tactile impulses there is an alternative path : they may pass up in the primary sensory neurons in the posterior columns of the same side or they may cross the spinal cord and pass up in the anterior ground bundle of the opposite side along a secondary sensory neuron (2). By the time that tactile impulses from the lower limbs have reached the upper cervical region of the cord it is probable they have completely crossed over to the opposite side. Impulses for passive position and tactile discrimination pass to the opposite side in the superior sensory decussation in the medulla (3). Non-sensory afferent impulses concerned in locomotion and preservation of equilibrium pass up the direct cerebellar tract (5) on the same side to the cerebellum.

What, then, is the sensory loss characteristic of tabes dorsalis and how does it compare with lesions of the posterior roots and of the spinal cord respectively ? In 1895 Max Laehr [12] in an admirable paper investigated the sensory changes in 60 tabetics. His conclusions are as follows : 1. Hyperæsthesia appears to be a regular and generally early sensory disturbance of tabes. 2. The sensory disturbances for a long time consist in diminished sensibility for light touch, whilst in the legs there is at the commencement diminution of painful sensibility and of the sense of position. The latter generally appears to precede the trunk hyperæsthesia. 3. The localisation in the trunk usually corresponds to the distribution of the middle or lower dorsal nerves. Their further distribution follows the encircling zone of the trunk and extends upwards and downwards in a characteristic manner to the arms. In 16 cases it spread to the arms, first to the axilla, then to the ulnar side of the arm, and lastly to the radial. 4. The extent of this tactile anæsthesia is quite characteristic ; it corresponds not to the area of distribution of the peripheral nerves but to that of the spinal roots and their intramedullary projection fibres. Chipault [13] in 1896 confirmed these results and noted the characteristic distribution of the sensory loss. Marinesco [14] in 1897 pointed out the preponderance of

[5] Redlich and Obersteiner: (Abstract) Neurologisches Centralblatt, 1894, p. 454.
[6] Orr and Rows: Brain, 1904, p. 461.
[7] Thomas and Hauser: Nouvelle Iconographie de la Salpétrière, 1902, p. 412.
[8] Marie: Maladies de la Moelle, Paris, 1892.
[9] Von Leyden and Goldscheider: Die Erkrankungen des Rückenmarks und der Medulla Oblongata, Wien, 1904.
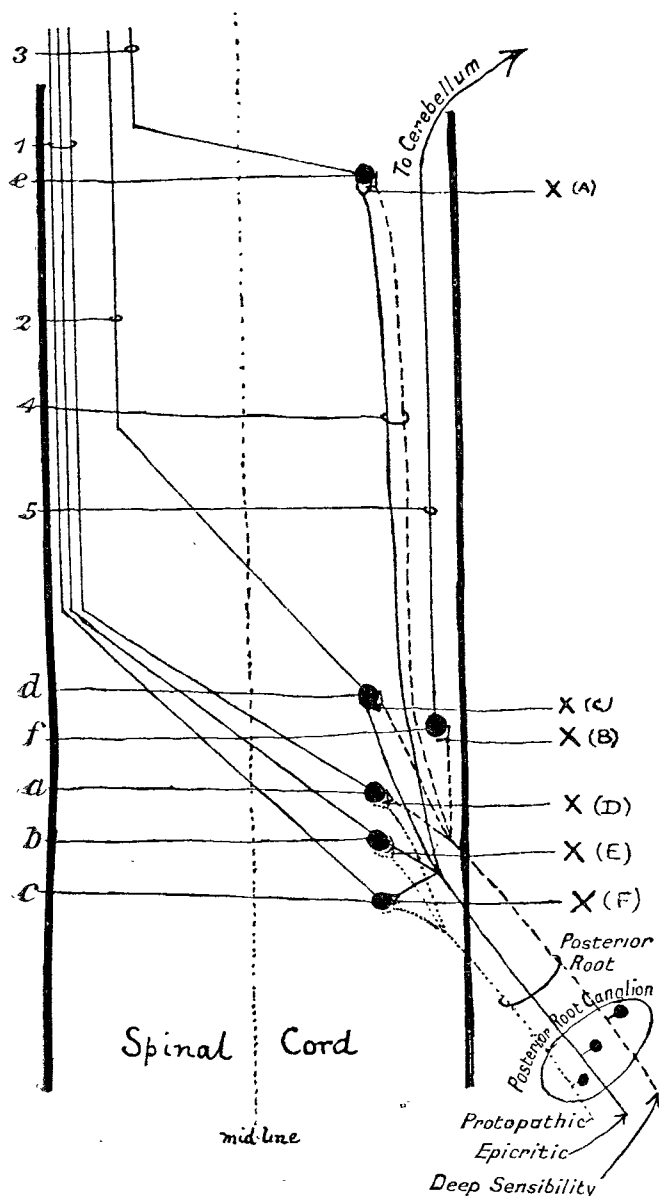[10] Head, Sherren, and Rivers: Brain, 1905, p. 116.
[11] Head and Thompson: Ibid., 1906, p. 537.

[12] Max Laehr: Archiv für Psychiatrie, 1895, vol. xxvii., p. 688-756.
[13] Chipault: Médecine Moderne, 1896, No. 44.
[14] Marinesco: La Semaine Médicale, 1897, No. 47.

FIG. 1.—DIAGRAM OF THE AFFERENT PROTO-NEURONS AND THEIR INTRAMEDULLARY TERMINATIONS.

a. Specific receptor for painful impulses ⎫
b.   ,,   ,,   ,,   heat   ,, ⎬ In the posterior horns.
c.   ,,   ,,   ,,   cold   ,, ⎪
d.   ,,   ,,   ,,   tactile   ,, ⎭

e. Specific receptor for impulses concerned with passive position and tactile discrimination ... ... } In the gracile and cuneate nuclei.

f. Specific receptor for non-sensory afferent impulses ... ... } In Clarke's column.

1. Sensory fibres of the second order for pain, heat, and cold ... ... } Part of Gowers's tract.

2. Sensory fibres of the second order for touch. ... ... } In anterior column.

3. Sensory fibres of the second order for passive position and tactile discrimination. ... ... } In superior sensory decussation.

4. Long fibres in the posterior column of cord.

5. Spino-cerebellar tracts for non-sensory afferent impulses.

X = Position of the initial lesion in tabes dorsalis.

painful and tactile loss over loss of thermal sensibility. Foerster and Frenkel [15] in 1900 investigated chiefly the relation of the ataxy to the sensory changes. They found no single case of tabetic ataxia without change in joint sensibility. With regard to the changes in skin sensibility, they found that analgesia was most common on the lower limbs, while tactile anæsthesia was more common in the trunk. Disturbances of heat and cold sensibility were rare. Mott [16] comes to very much the same conclusion, except that

he states that he has met with several cases of ataxy without loss of position at the joints.

The conclusions arrived at by Dr. Head and myself are largely in accordance with those of Laehr and may be summarised as follows : 1. Loss of painful sensibility frequently exists without thermo-anæsthesia. This occurred in the case described below. While analgesia is comparatively frequently met with in cases of tabes, disturbances of sensibility to heat and to cold are rare. 2. In cases where thermo-anæsthesia does exist the loss of sensibility to heat may be separate from loss of sensibility to cold. 3. When the area of cutaneous analgesia is widespread and persistent it is accompanied by diminution or loss of the pain of excessive pressure. This is well seen in the case of tabes with unilateral analgesia quoted below. Over the right leg sensibility to the pain of a pin prick was present, while over the left leg it was lost. Observations with the algometer showed that the pain of deep pressure was normal in the right leg, while it was absent in the left. 4. Light tactile anæsthesia is often accompanied by a diminution of the sensibility to pressure. The case quoted also illustrates this point. 5. Sense of position and movement may be completely lost in a limb and yet pressure may be easily recognised and localised. This fact must be familiar to everyone who has investigated any number of tabetics and is one of the most characteristic forms of dissociated sensory loss in this disease. 6. Ataxia may exist without any change in the sense of passive position or movement and without loss of cutaneous sensibility, and must then be due to the interruption of non-sensory afferent impulses. On the other hand, loss of the sense of passive position is always accompanied by a greater or less degree of ataxy. 7. The cutaneous analgesia in tabes dorsalis spreads in a segmental manner and closely resembles the distribution of the spread of the loss of cutaneous pain in syringomyelia.

From these facts it is clear : (A) That the sensory changes in tabes dorsalis are not due to changes in peripheral nerves ; for their area of distribution corresponds to that of the spinal roots and their intramedullary prolongations and not to the distribution of the peripheral nerves. (B) That the sensory changes in tabes are not due to a coarse lesion of the posterior roots. In tabes it is not protopathic or epicritic sensibility which is lost. The anæsthesia is of a fundamentally different character. Thus cutaneous pain may be lost without affection of thermal sensibility, a condition incompatible with loss of protopathic sensibility. Tactile anæsthesia may exist without loss of the power of discriminating minor degrees of heat and cold, which is incompatible with loss of epicritic sensibility. More important still, we may have complete loss of the sense of position in a limb with retention of the pressure sense, a condition which is never found in lesions of the deep system of nerves. Hence it is clear that the sensory changes in tabes are not produced by lesions of the deep, epicritic, and protopathic fibres as they pass into the cord in the posterior roots. (C) On comparing the sensory changes found in tabes with those due to lesions of the secondary sensory system it will at once be seen how close is the similarity between them. The separation of painful from thermal sensibility, the dissociation of the sense of passive position from tactile sensibility, are found both in tabes and in intramedullary lesions from various causes. The separation of the pain of deep pressure from the recognition of the pressure itself and its association with the loss of cutaneous pain are characteristic of both tabes and lesions of the secondary sensory neurons. Consequently we must conclude that in tabes the sensory loss is similar in character to that met with in lesions of the secondary sensory system

III. *What is the bearing of these observations upon the origin of the tabetic process?*—Although on investigating the loss of sensibility in tabes it was found to correspond to that due to lesions of the secondary sensory system, yet it is well known that in tabes the primary sensory neurons are alone affected. In the case of tabes dorsalis of which a summary is appended, the anæsthesia present was of the character described and the post-mortem examination showed that pathological changes existed in the fibres of the primary sensory neurons alone. We can therefore only explain these sensory changes on the supposition that the primary change in tabes begins in the synaptic junctions between the primary and secondary sensory neurons. Thus the lesion of tabes must be considered as a degeneration of the

15 Foerster and Frenkel : Archiv für Psychiatrie und Nervenkrankheiten, Band 33, Heft I., S. 109 and 450.
16 Mott : Tabes in Hospital and Asylum Practice, London, p. 60.

pericellular networks around the cells of the secondary sensory neurons. If reference be made to Fig. 1 it will be seen that a lesion at X (A) would produce loss of the sense of passive position, of active movement and of tactile discrimination. This would of itself produce some ataxy of the limb affected. A lesion at X (B) might produce ataxy without loss of cutaneous or muscular sensibility. Marked ataxy may be dependent on the loss of non-sensory afferent impulses, while all sensory afferent impulses are intact. A lesion at X (C) would produce tactile anæsthesia, both light touch and pressure being affected simultaneously. A lesion at X (D) would produce cutaneous analgesia and at the same time a loss of the pain of deep pressure. A lesion at X (E) would produce loss of sensibility to heat, while at X (F) a lesion would produce loss of sensibility to cold. These lesions may be present either singly or in combination and will account for all the diverse sensory changes found in tabes dorsalis. From the known affection of the posterior columns and posterior roots it might have been expected that the sensory changes in tabes would correspond to the peripheral type ; but, as we have shown, they resemble in character those produced by lesions of the secondary sensory system.

The lesion at the synaptic junctions between the primary and secondary sensory neurons well explains how it is possible that pain may be affected alone, heat affected alone, or cold affected alone, and how in other cases these conditions may be combined. Such a lesion also would explain why loss of the sense of passive position may occur without any loss of the pressure sense. A gradual affection of the first synaptic junction would also give an explanation of a marked characteristic of the sensory disturbances of tabes— namely, delay in response to stimulation, which is more often present in this disease than in any other sensory affection. Thus on its afferent side tabes dorsalis may be defined as a lesion of the synaptic junctions between the primary and secondary sensory neurons.

Does this view accord with the most modern pathological work on tabes dorsalis? The above hypothesis assumes that the primary lesion is in the pericellular networks around the cells of the secondary sensory neurons, followed by a slow atrophic process in the axons of the afferent protoneurons, which extends outwards along the posterior roots as far as the posterior root ganglia. Recent work on the pathological anatomy of tabes dorsalis has been summed up by Williamson [17] as follows. The changes in the intramedullary fibres of the primary sensory neuron (spinal cord changes) are well marked : changes in the cells of the posterior ganglion are often slight or absent ; those in the peripheral fibres (peripheral nerves) are very slight and limited to the termination of these nerves. The changes in the peripheral nerves are disproportionate to those of the fibres within the cord. At an early stage the changes in the intramedullary fibres of the posterior nerve roots are well marked, whilst the extramedullary fibres of the posterior roots outside the pia mater are normal or show only very slight changes. Thus Williamson [18] has shown that in a case of early tabes with well-marked sclerosis of the posterior columns the posterior roots were normal, and Erb, himself a supporter of the radicular theory, states that it is certainly remarkable that the degeneration of the posterior roots is often much less advanced and less intense than that of their intramedullary prolongations.

Thus all recent work tends to show that the earliest lesion in tabes lies within the spinal cord. On pathological grounds it is unlikely that this intramedullary degeneration is caused by meningeal thickening around the posterior roots at their entrance to the spinal cord. The meningitis which this view postulates is more frequently absent than present (Ferrier), and, when it exists, is more often of a character of a secondary thickening than of an inflammatory process. Moreover, meningitis, syphilitic or otherwise, when it does occur, is not a common cause of intraspinal degeneration. The affection of the intramedullary prolongations of the posterior roots is a selective one and not likely to be induced by a coarse meningeal lesion at the entrance of the roots into the cord. Thus the fine posterior root fibres, which enter the zone of Lissauer, often escape degeneration when it is already well marked in Burdach's column. These considerations indicate that meningeal thickening cannot be

a causative factor. Lastly, Spielmayer,[19] using Cajal's new axis-cylinder staining method, concludes that "there is in tabes a marked atrophy of the central terminations of the sensory protoneurons and that this atrophy is especially marked in the pericellular networks around the cells of Clarke's column and those of the posterior horn." Consequently it may fairly be said that recent pathological research is not inconsistent with the view here set forth and arrived at on purely clinical grounds, that tabes dorsalis begins as a lesion of the synaptic junctions between the primary and the secondary sensory neurons.

The following case of tabes dorsalis is interesting in view of the fact that the anæsthesia was unilateral and that it was possible to compare the condition of the anæsthetic leg with one of normal sensibility. Moreover, the psycho-physical examinations were made under favourable conditions and an opportunity was afforded of examining the spinal cord shortly afterwards, the patient dying from heart failure the result of syphilitic mes-aortitis. The following is a summary of the case :—

The patient was a female, aged 51 years. She had suffered for 15 years with lightning pains. In June, 1906, she noticed that she could not use the left leg properly and that the left leg seemed to be numb. For three years she had had trouble with micturition and defæcation. She occasionally had incontinence and she could never feel the passage of urine or fæces. She had been very short of breath for three months. She had been married 22 years and had had no children and no miscarriages. No direct history of syphilis was obtained. The patient was admitted to Poplar Hospital on Feb. 7th, 1907, and she died on June 4th of the same year. The observations on the state of her sensibility were made in February and March, when her general condition was excellent. Towards the end of March an attack of cardiac failure supervened and œdema made accurate observations of the sensibility of the legs from this time impossible. Her mental condition was very intelligent and alert ; she answered well to all control tests. With regard to motion, there was no loss of power. Electrical reactions were everywhere normal. Ataxia was much more marked in the left leg than on the right. Sensation.—There were shooting pains in the arms and legs, not more marked in one leg than the other. Girdle pains were very severe and lasting for two hours at the level of the tenth and eleventh thoracic spines. There was insensibility [20] to pain of a pin prick over the left lower limb (see Fig. 2). The upper limit was sharply

FIG. 2.
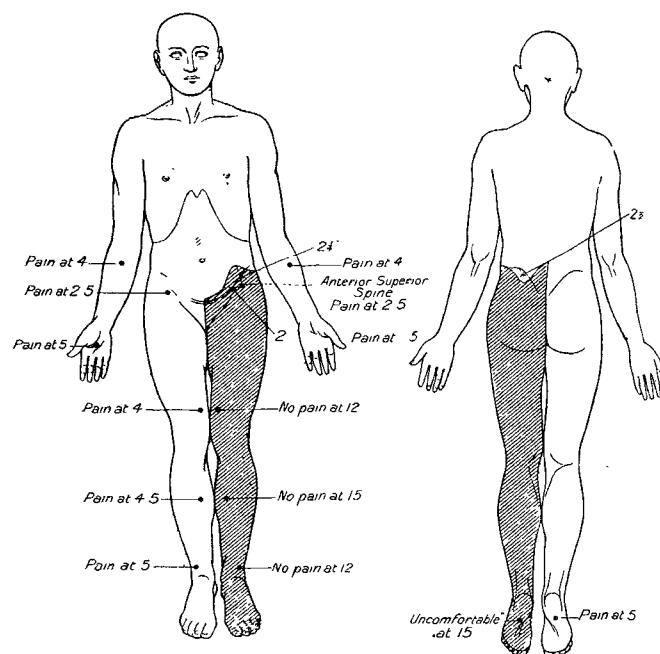
defined and never varied and corresponded exactly to the caudal border of the eleventh thoracic segment. Insensibility to the pain of a powerful faradic current was present

17 Williamson: Diseases of the Spinal Cord, London, 1908, p. 333.
18 Williamson : Op. cit., p. 332.
19 Spielmeyer : Archiv für Psychiatrie und Nervenkrankheiten, 1905, Band xl., p. 389.
20 Such absolute analgesia is not common in tabes dorsalis ; generally sensibility is merely diminished for a long period before it becomes completely lost.

over the same area. The pain of deep pressure was tested by means of the algometer. There was complete loss of deep pressure pain over the left lower limb, although the pressure itself and its gradual increase were appreciated.

Algometer Readings in Kilogrammes.

—	Right.	Left.
Sole	Pain at 5·0	" Uncomfortable " at 15.0
External malleolus...	,, 5·0	No pain at 12·0
Shin	,, 4·5	,, 15·0
Inner side of the knee	,, 4·0	,, 12·0
Anterior superior spine of the ilium	,, 2·5	Pain at 2·5
Forearms	,, 4·0	,, 4·0
Hands...	,, 5·0	,, 5·0

With regard to thermal sensibility there was no loss anywhere. Intermediate degrees of heat and cold could be everywhere discriminated. Light touch as tested by cotton wool was absent over the left leg and diminished over the left thigh. When tested with von Frey's hairs: On the face and hands No. 5 and No. 8 at once appreciated. On the right leg No. 5 and No. 8 at once appreciated. Over the left foot and leg there was no response to No. 5 or to No. 8. Over the left thigh there was no response to No. 5; No. 8 called a touch. Pressure was diminished over the left foot and left leg, elsewhere it was normal. Localisation of touch was accurate over the right leg. No attempt was made to localise at touch on the left leg and left foot; localisation was faulty over the left thigh. Sense of passive position was absent in the left great toe and left ankle-joints. It was present elsewhere. The following are records of answers.

—	Right leg.	Left leg.
Great toe. Extension.	10 right.	1 no reply ; 9 wrong.
Flexion.	10 right.	10 right.
Ankle. Extension.	10 right.	1 doubtful ; 9 right.
Flexion.	10 right.	2 doubtful ; 4 right ; 4 wrong.
Knee. Extension.	10 right.	10 right.
Flexion.	10 right.	10 right.

Movement of the left great toe was not recognised till an angle of 45° had been traversed. In the left ankle movement was not recognised till an angle of 60° had been traversed. On the right side movement was recognised almost at once. The difference between the two sides was most striking. Compass test: "Tactile discrimination." The following results were obtained on Feb. 22nd, 1907, and were confirmed on subsequent occasions.

Right.			Left.	
Palms of hands. 3 cm. apart.	1. 10 right. 2. 10 right.	...... 2·5 cm. apart.	1. 10 right. 2. 10 right.
Soles of feet. 3 cm. apart.	1. 9 right; 1 wrong. 2. 9 right; 1 wrong.	......	No attempt to discriminate one point from two points.
Outer side of leg. 10 cm. apart.	1. 10 right. 2. 8 right; 2 wrong.	...... 20 cm. and 30 cm. apart.	1. 10 right. 2. 10 wrong.

This shows that tactile discrimination was lost over the left leg and left foot. With regard to the cranial nerves the pupils were small and equal. They reacted to accommodation but not to light. The ocular movements were normal. There was no nystagmus. Vision was good. The optic discs were normal. The other cranial nerves were normal. The knee-jerks were absent on both sides. The plantar reflex gave a flexor response. Ankle clonus was not obtained.

The joints, the skin, and the nails were normal. There was difficulty in holding the urine and anæsthesia of the sphincters existed. There was well-marked aortic regurgitation with much dilatation and hypertrophy of the left ventricle. From March onwards the patient suffered with severe attacks of cardiac pain, in one of which she finally died.

Necropsy.—A post-mortem examination was performed the day after death. There was general œdema. The heart showed dilatation and hypertrophy of the left ventricle with incompetent aortic valves. There was very marked atheroma, with much scarring of the wall of the aorta. The liver was enlarged and congested. The kidneys were healthy. As to the brain, the dura mater was normal. The pia arachnoid was transparent and not thickened ; it stripped easily and was not adherent to the cortex. There was no atrophy of the convolutions. There were no naked-eye changes in the cerebrum, cerebellum, basal ganglia, or pons. With regard to the spinal cord, there was slight flattening on the posterior surface. Microscopic sections at the level of the first sacral segment showed marked degeneration of posterior columns when stained by Weigert's method. The cornu commissural tract was not affected. Marchi's method showed degenerated fibres on both sides. Section through various levels of the lumbar and thoracic cord gave similar results. A microphotograph at the level of the third thoracic segment stained by the Weigert-Pal method is shown. (Fig. 3.) Sections

FIG. 3.
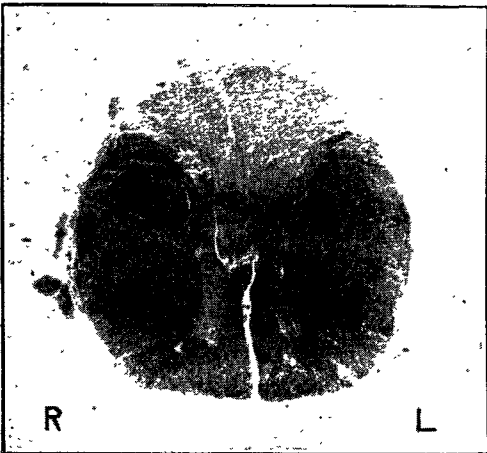
Section at the level of the third thoracic segment stained by the Weigert-Pal method. Much degeneration of the exogenous tracts of the posterior columns.

through the *cervical* cord at various levels show, when stained by Marchi's method, marked degeneration on the right side and slight degeneration on the left side. A microphotograph of a section at the level of the seventh cervical segment stained by Marchi's method is shown. (Fig. 4.)

FIG. 4.
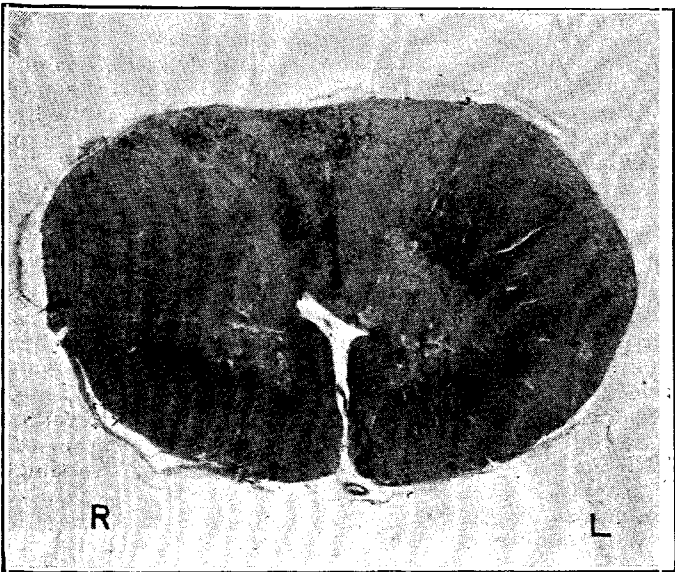
Section at the level of the seventh cervical segment stained by Marchi's osmic acid method. The section shows many recently degenerated fibres in the right posterior column, very few in the left posterior column.

Stained by the Weigert-Pal method the section shows a greater degeneration in the left column of Goll than in the right column of Goll. This indicates that the degeneration of the left column of Goll was of long

standing while much of that of the right column of Goll was of recent date. Serial sections were made of the medulla and the recently degenerated fibres in the right column of Goll could be traced upwards to the right nucleus gracilis where they could be seen to end. Sections of the upper part of the medulla, of the pons, and through the region of the corpora quadrigemina were made and stained by Weigert's method and by Marchi's method. Nothing abnormal was detected. Sections of the posterior roots were made below and above the twelfth thoracic segment. No difference could be observed between the two sides. As to the posterior root ganglia, these were stained by Nissl's methylene blue method. No evidence of gross changes in the nerve cells were observed.

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HYDROCHLORIC ACID IN THE GASTRIC CONTENTS IN CANCER :

A REPLY TO PROFESSOR B. MOORE.

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IN a paper entitled, "Variations in the Free Hydrochloric Acid of the Gastric Contents in Cancer and the so-called ' Physiologically Active' Hydrochloric Acid,"[1] Professor B. Moore criticises a paper recently published by us[2] in which we had occasion to comment on some previous work by himself and certain of his colleagues. We had hoped that our comments were sufficiently intelligible to obviate the necessity for further explanation, but Professor Moore is evidently of a different opinion and has published a lengthy " criticism " in answer to our remarks.

In the first place we feel it incumbent upon us to point out that Professor Moore misquotes the title of our paper more than once in his criticisms and so, doubtless unintentionally, conveys to his readers an entirely erroneous impression as to its scope. Our paper was entitled, " A Study of the Variation in the Secretion of Hydrochloric Acid in the Gastric Contents of Mice and Rats as Compared with the Human Subject in Cancer," *not*, as Professor Moore puts it, " On the Acidity of the Gastric Contents of Mice and Rats with Transplanted Tumours and also of a Number of Cases in Man." The difference is vital, for although as a matter of fact in all our experiments we made determinations of the total acidity we did not profess to regard that series of estimations as of any great importance, but merely as having a subsidiary interest in connexion with the entirely independent determinations of physiologically active hydrochloric acid (and incidentally of free hydrochloric acid) which constituted the main object of our research.

Professor Moore next quotes a statement of ours to the effect that the *conclusions* at which we have arrived contradict or materially modify those arrived at by himself and others, and adds as a criticism of this statement that he desires no better confirmation of his *results* than is given in our table when using his methods "which are those established by many previous observers." Here again he conveys a somewhat erroneous impression, for although our conclusions, in certain respects, greatly differ from those of Professor Moore we have nowhere disputed his results, nor his well-known ability as a chemist, nor the accuracy of the methyl-acetate process as employed by him for estimating the free hydrochloric acid in the gastric contents. But inasmuch as in his first paper[3] he finds as an average in 12 cases of cancer 0·0039 per cent. of free hydrochloric acid in the gastric contents, while in his second paper[4] he finds as an average of 13 cases of cancer 0·0515 per cent., or nearly 13 times as much, and in the same paper demonstrates an average of 0·063 per cent. in 20

1 Bio-chemical Journal, vol. iii., No. 10, 1908.
2 Proceedings of the Royal Society, B. vol. lxxx., 1908. A preliminary note on our investigations appeared in THE LANCET of Nov. 10th, 1906, p. 1276.
3 Proceedings of the Royal Society, March, 1905.
4 Bio-chemical Journal, May, 1906.

non-malignant cases, we pointed out as an obvious inference that no stress can be laid on, nor deduction of any kind be drawn from, such widely divergent results. This conclusion, moreover, applies equally to the work of Dr. A. S. Morton Palmer[5] who finds, as an average of 14 cases, 0·0217 per cent. of free hydrochloric acid and to our own average of 0·0407 per cent. in 13 cases of cancer, although attention may be called to the fact that our average agrees fairly well with that recorded in Professor Moore's second paper.

As an explanation of the causes of these differences in the amounts of free hydrochloric acid found in the gastric contents the simplest appears to be, as we suggested, either dilution of the test meals with water during withdrawal or the varying intervals (from an hour to two hours) at which the test meals were withdrawn ; but Professor Moore indignantly repudiates the first suggestion and disputes the second, notwithstanding his own statements as to variation in time of withdrawal. He is even inclined to be sarcastic at our expense and refers in his criticism to "the enormous difficulty experienced by Copeman and Hake" in the withdrawal of the test meals. This is not quite fair comment on his part, for in our paper (p. 456) we discuss this matter at length and show that we relied on experts at the Middlesex and Westminster Hospitals and at the Cancer Hospital, Brompton, as regards administration and withdrawal of the test meals, and we point out that it was owing to the remarkable variation of results in the estimations of both free and physiologically active hydrochloric acid that we made special inquiries and found that water had been added in certain cases owing to the difficulty experienced by the experts themselves. We further point out that only by great insistence and by special arrangement with Mr. C. H. Leaf and his house-surgeon (Mr. Allan) at the Cancer Hospital were we able to obtain undiluted test meals, and we actually quote Mr. Allan, who informed us that "where test meals had been given, two out of three cases had to be abandoned owing to the impossibility of inducing the withdrawal of the fluid from the stomach of the patient." Moreover, Dr. Morton Palmer himself calls particular attention to the importance of avoiding the use of water in the withdrawal. We are of opinion that not a few interesting instances of absence or marked diminution of free hydrochloric acid in the gastric contents, recently so frequently recorded, may not improbably owe their origin to a neglect of this vital rule, so strongly insisted on by Ewald, who originally suggested this aid to diagnosis.

In the face of the wide divergences in the percentage of " free " hydrochloric acid in the gastric contents found by Professor Moore and others, above quoted, and for other reasons specially discussed in our paper, it seemed to us more logical to estimate what has been termed the "physiologically active" hydrochloric acid, and we are of opinion that Lüttke in the first instance, and Dr. W. H. Willcox later, have made out an incontrovertible case for this procedure. Both of these workers regarded the estimation of free hydrochloric acid as involving a distinct fallacy and it is therefore somewhat surprising to find Professor Moore quoting Dr. Willcox's paper[6] in support of his own views, since in this particular paper Dr. Willcox is at great pains to expose the fallacy above alluded to, and we have referred to the point in our paper. Moreover, Dr. Willcox has not altered his views, since in a paper published in THE LANCET[7] in 1908 he says :—

As I have pointed out, it is absurd to draw a deduction between free HCl and HCl combined with proteid ; therefore the presence or absence of free HCl is of no value as evidence. The important question is, "What is the amount of active HCl present ?"

Here it is obvious, in fact, that Professor Moore and ourselves join issue, for in his criticism of our paper he calls "physiologically active" hydrochloric acid "a misnomer" and considers that the method "shows something quite different from hydrochloric acid in any active form." We think that it is neither necessary to repeat the lengthy arguments already put forward by us in support of our view of the question, nor does it serve any useful purpose, inasmuch as Professor Moore refuses to recognise the value of the estimations in the face of reliable scientific authority. To emphasise the apparent uselessness of pursuing the argument Professor

5 Guy's Hospital Reports, 1906.
6 THE LANCET, June 10th, 1905, p. 1566.
7 THE LANCET, July 25th, 1908, p. 221.